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Comparison of the effect of electroconvulsive therapy performed by administering propofol with and without rocuronium

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Abstract
Aim: In this study, we aimed to compare groups in terms of cardiovascular changes and side effects by administering propofol to patients in the control group and propofol and rocuronium bromide to patients in the study group during electroconvulsive therapy.
Material and Methods: Eighty-four patients with ASA I-II schizophrenia, major depression and bipolar disease were included in the study. Heart rate, MAP (mmHg) and ECG of the patients were recorded both before induction and at the end of convulsion. Patients in the R group (n = 42) were administered with 2% lidocaine IV 0,5 mg / kg, propofol IV 1-2 mg / kg and rocuronium IV 0,2 mg / kg for induction. Patients in the P group (n = 42) were given 2% lidocaine IV 0,5 mg / kg and propofol IV 1-2 mg / kg only for induction.
Results: Pd values before induction were statistically lower than Pd values after ECT in both P and R groups. In both patients in the P group and the R group, the post-ECT values for HR were statistically higher than the pre-induction values. Pre-induction MAP values were statistically lower than post-ECT MAP values in both P and R groups.
Discussion: In ECT, when we compare the application of propofol and rocuronium with the application of propofol without muscle relaxants, propofol as an anesthetic agent provided an adequate and safe anesthesia with rapid recovery and a minimal side effect profile except for postoperative temporary myalgia in these patients.

Keywords
ECT, Propofol, Rocuronium

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Introduction

Electroconvulsive therapy (ECT) is a procedure in which electrical stimulation is applied to the scalp to induce a generalized seizure [1]. Muscle relaxation is commonly used to prevent injuries associated with ECT-induced tonic-clonic seizures. Hypnotics are administered to induce amnesia and unconsciousness, so that patients do not notice the period of muscle relaxation and generalized seizures [2].

Propofol is commonly used because it provides the short-term loss of consciousness required for ECT [3]. Neuromuscular blockade is important for the safe use of ECT so as to protect the patient from dental, tongue, and musculoskeletal trauma [4]. Administration of rocuronium as a neuromuscular blocker for ECT anesthesia is attracting more attention as sugammadex is increasingly used in anesthesia practice. Sugammadex is a new generation reversal agent used to terminate the action of nondepolarizing neuromuscular blockers (vecuronium and rocuronium) [5,6].

Many ECT changes occur during the ECT procedure. Apart from convulsions, ECT also causes parasympathetic discharge. Depending on the vagal and sympathetic effects of ECT, side effects may include bradycardia, tachycardia, atrial-ventricular arrhythmias, and S-T and T-wave changes [7].

This study aims to compare the groups in terms of cardiovascular changes and side effects by administering propofol to the patients in the control group and propofol and rocuronium bromide to the patients in the study group during the induction of anesthesia.

Material and Methods

This study was conducted on patients who underwent ECT between 01/06/2018 and 01/01/2020 after approval of the Local Ethics Committee. Patients diagnosed with severe and treatment-resistant depression, psychosis, bipolar disorder, and schizophrenia and scheduled for an ECT were included in the study. The study was conducted in accordance with the Declaration of Helsinki of the World Medical Association published in 2013.

We studied patients with an American Society of Anesthesiologists (ASA) score I-II, aged 18-65 years, with a body mass index (BMI) between 18-30 kg/m2. Patients with a history of pregnancy, a history of epilepsy, a history of cardiovascular diseases, those with propofol-rocuronium-sugammadex allergy, patients in the ASA III-IV category, and patients not wishing to be enrolled in the study were excluded from the study.

Patients were not pre-medicated and they were asked to avoid food and water for at least 8 hours before induction. In the ECT room of the psychiatric ward, all patients had a 12-lead ECG, a mean arterial blood pressure (MAP) reading and peripheral oxygen saturation (SpO2) measured (Massimo-Radical 7) by pulse oximetry.

Age, sex, ASA score, body mass index (kg/m2), HR (beats/minute), SpO2 and MAP were recorded before induction. In addition, patients had an ECG before the procedure. When the "RAMSAY sedation scale" reached the 5-6 level, patients were stimulated with an ECT device [8].

A total of 84 patients were recruited, of whom 42 received ECT with propofol and rocuronium (group R) and 42 received ECT with propofol alone (group P). Group R was administered 0.5 mg/kg lidocaine (Aritmal 2% I.M./I.V./S.C. 100mg/5cc, Osel İlaç Sanayi ve Ticaret A.Ş.), 1 mg/kg propofol (Propofol-Lipuro 1%, 200mg/20ml, B. Braun Medikal Dış Ticaret A.Ş. Esenler/İSTANBUL), 0.2 mg/kg rocuronium (Esmeron® Vial 50mg/5ml, Merck Sharp Dohme İlaçları Ltd. Şti., Levent/İstanbul), and 2mg/kg sugammadex (BRIDION 200 mg/2ml, IV vial, Merck Sharp Dohme İlaçları Ltd. Şti. Levent/İSTANBUL) for decurarization, while group P was given 0.5 mg/kg lidocaine and 1 mg/kg propofol for sedation purposes. ECT was administered using the SPECTRUM 5000Q device (MECTACorporation), which generates a constant current between 0.9 amps and 500 ohms with intermittent, bidirectional square wave stimulation. As soon as the convulsions stopped, patients had a repeat ECG.

HR, SpO2 and MAP were recorded at the end of convulsions. The time from the initiation of bitemporal electroshock to the end of convulsions was recorded as convulsion length (seconds). Sugammadex IV 2mg/kg was administered to Group R after the convulsion ended. The time from the end of ECT to the patient's response to verbal stimuli was considered the recovery time (seconds). Patients with a modified Aldrete score of 9-10 were sent to their rooms after some time in the observation room [9]. Nausea, vomiting, hypotension, hypertension, headache, tachycardia, bradycardia, myalgia, allergic reactions and arrhythmias observed in patients within the first 24 hours after ECT at the psychiatric ward were recorded. Metpamide 10 mg I.V. was administered to patients with complaints of nausea and vomiting. Patients with myalgia were given paracetamol 500 mg I.V.

Statistical Analysis

The minimum number of subjects required for the P measurement to be significant for a difference of 6.4 ± 6.8 in females was set at 15, and the minimum number of subjects required for a difference of 7.2 ± 6.2 units to be significant in males was set at 10 ($\alpha = 0.05$, $1-\beta = 0.90$). The analysis was performed with Gpower version 3.1.

Descriptive statistics of the data obtained from the study were expressed with mean and standard deviation values for continuous variables and with frequency and percentage analysis for categorical variables. The paired t-test was used to compare the pretest-posttest measurements of the study groups, while the independent samples t-test was used to compare these values by study group. The chi-square test was used to compare differences between categorical variables. The independent samples t-test was used to compare convulsion lengths and recovery times by study group and sex. In addition, the relationships between continuous variables were examined with the Pearson correlation analysis. Analyses were performed using the SPSS 22.0. $p<0.05$ was considered significant.

Results

The mean age was 37.14 ± 10.9 years in group P and 37.98 ± 12.07 years in group R, and there was no statistically significant difference between the groups. The mean body mass index was 25.14 ± 2.89 years in P group and 25.05 ± 3.04 in R group, and there was no statistically significant difference between the groups. There was no statistically significant difference between the P group and the R group in terms of convulsion

Table 1. Demographic Data

	Group P (n=42)	Group R (n=42)	p
Age (years) (Mean± SD)	37.14±10.9	37.98±12.07	>0.05
BMI (kg/m2) (Mean± SD)	25.14±2.89	25.05±3.04	>0.05
Sex (F/M) (n)	23/19	23/19	>0.05
Convulsion Length (sec)	30.14±2.93	29.33±2.68	0.191
Recovery Time (sec)	372.38±25.82	389.36±18.40	0.001*

SD: Standard Deviation, BMI: body mass index, F: Female, M: Male, p<0.05 statistically significant

Table 2. Comparison of P Group and R Group in terms of Heart Rate (HR), Mean Arterial Pressure (MAP) and Peripheral Oxygen Saturation (SpO2)

	Group P (n=42)	Group R (n=42)	p
HR-Before (beats /min (Mean±SD)	75.48±5.64	75.60±5.31	0.921
HR-After (beats /min) (Mean±SD)	92.00±8.23	89.10±7.88	0.102
MAP-Before (mmHg) (Mean±SD)	77.00±4.83	76.69±4.78	0.768
MAP-After (mmHg) (Mean±SD)	87.38±6.71	88.60±6.62	0.406
SpO2-Before (%) (Mean±SD)	98.93±0.84	99.10±0.66	0.313
SpO2-S (%) (Mean±SD)	97.31±0.92	97.52±0.80	0.260

HR-BEFORE: Heart rate before convulsion, HR-AFTER: Heart rate after convulsion, MAP-BEFORE: Mean arterial pressure before convulsion, MAP-AFTER: Mean arterial pressure after convulsion, SpO2-BEFORE: Peripheral oxygen saturation before convulsion, SpO2-AFTER: Peripheral oxygen saturation after convulsion, Mean ±SD: Mean standard deviation, p<0.05 statistically significant

Table 3. Comparison of ECG Wave Duration (sec) between P Group and R Group

	Group P (n=42)	Group R (n=42)	p
P-BEFORE (msec) (Mean±SD)	0.15±0.02	0.15±0.02	0.912
P-AFTER (msec) (Mean±SD)	0.15±0.02	0.15±0.02	0.914
Pd-BEFORE (msec) (Mean±SD)	18.43±2.46	18.62±2.27	0.712
Pd-AFTER (msec) (Mean±SD)	20.05±2.48	20.12±2.33	0.892
QRS-BEFORE (sec) (Mean±SD)	0.08±0.01	0.08±0.01	0.149
QRS-AFTER (msec) (Mean±SD)	0.08±0.01	0.08±0.01	0.127
QT-BEFORE (msec) (Mean±SD)	0.30±0.04	0.29±0.03	0.511
QT-AFTER (msec) (Mean±SD)	0.30±0.04	0.29±0.03	0.471

P-BEFORE: P wave duration before convulsion, P-AFTER: P wave duration after convulsion, Pd-BEFORE: P wave dispersion before convulsion, Pd-AFTER: P wave dispersion after convulsion, QRS-BEFORE: QRS wave duration before convulsion, QRS-AFTER: QRS wave duration after convulsion, QT-BEFORE: QT interval duration before convulsion, QT-AFTER: QT interval duration after convulsion, Mean± SD: Mean standard deviation, p<0.05 statistically significant

length (CL) (p > 0.05). However, it was found that the recovery time (RT) was statistically significantly lower in the P group than in the R group (p = 0.001) (Table 1).

There was no statistically significant difference between P group and R group in terms of HR values (p > 0.05). In addition, there was no statistically significant difference between P group and R group in terms of MAP values (p > 0.05). Furthermore, there was no statistically significant difference between P group and R group in terms of SpO2 values (p>0.05) (Table 2).

There was no statistically significant difference between P group and R group in terms of P-wave duration, P-wave dispersion, QRS wave duration and QT interval duration both in pre- and post-convulsion periods (p > 0.05) (Table 3).

Myalgia was observed in 21.43% (n=18), nausea/vomiting in 21.43% (n=18), tachycardia in 16.67% (n=14) and hypertension in 4.76% (n=4) of the 84 patients included in the study. There was no statistically significant difference between the two groups in terms of nausea/vomiting (p > 0.05). The number of patients with myalgia was statistically significantly higher in P group compared to R group (p = 0.008). It was statistically significantly more common in female patients than in male patients (p = 0.006).

Discussion

In this retrospective clinical study, which included 84 patients who were administered ECT, we compared the cardiovascular parameters (HR, MAP, waves measured by ECG) and peripheral oxygen saturation (SpO2) as well as side effects in ECT administered with both propofol and rocuronium (group R) or propofol alone (group P) for anesthesia management.

Prochnicki et al. studied the changes in QT interval and troponin T brought on by ECT in psychiatric patients and performed 12-lead ECG on all patients before premedication and 1 hour after ECT. Likewise, troponin T levels were checked twice: before ECT and 6 hours after ECT [10]. As a result, similar to our study, no significant difference was found in QRS width and QT interval in the ECT series.

In the present study, there was no statistically significant difference between P group and R group in terms of the duration of P wave before and after convulsion. P wave dispersion over 40 msec carries the risk of atrial arrhythmia [11]. In our study, no P-wave dispersion exceeding 40 msec was observed in any of the patients. In their study, Mızrak et al. investigated ECT effects on the ECG parameters of male and female patients. There was no statistically significant difference between the groups in terms of P-wave duration and P-wave dispersion [7]. In our study, there was no clinically threatening situation, since the Pd value was within normal range (<40 msec).

Takada et al. studied the effect of ECT on the cardiovascular system in patients over 50 years of age. A total of 38 patients aged 50-83 years, including 18 males and 20 females, without a systemic disease, were enrolled in the study. They showed no serious adverse effects and electroconvulsive therapy did not trigger any malignant arrhythmias or ischemia [12]. Locala et al. evaluated 110 ECT treatments with methohexital alone or combined with remifentanil. They found that systolic blood pressure was significantly lower at one minute following the end of seizure and five minutes after end of seizure in the remifentanil group [13]. In our study, blood pressure and HR were found to be statistically significantly higher after ECT compared to the same before induction in the present study.

Vishne et al. applied two protocols: Propofol (1 mg/kg) in 10 patients and propofol (0.5 mg/kg) + remifentanil (1 µg/kg) in 11 patients. All patients received 0.5-0.75 mg/kg IV succinylcholine as a muscle relaxant. They indicated that DAP and SAP were increased after convulsion compared to the pre-induction levels in both protocols [14]. Moacyr et al. included 30 patients in their study. Etomidate 0.15-0.3 mg/kg was administered to 10 patients, propofol 1-1.5 mg/kg to 10 patients, and thiopental 2-3 mg/kg to 10 patients, divided into 3 separate groups. All

patients received 0.5-1.25 mg/kg IV succinylcholine as a muscle relaxant. They found that pre-induction values of MAP were statistically significantly lower than post-convulsion values in all patients [15]. In this study, pre-ECT MAP values were found to be statistically significantly lower than MAP values measured after convulsions, in both groups P and R. It was assumed that an increase in MAP was observed as a result of the increase in catecholamines during ECT.

In our study, it was observed that there was no statistically significant difference between P group and R group in terms of convulsion length, and the recovery time was statistically significantly longer in R group than in P group. On the other hand, Algül et al. compared recovery times between the groups in their study and found that the propofol group had a significantly shorter recovery time than the propofol + remifentanil group. In our study, recovery time was statistically shorter in P group than in R group [13]. When using sugammadex (2-4 mg/kg), the fastest recovery of muscle strength was reported to be in about 1 minute, with an average of 1-3 minutes [16].

Generalized myalgia is a common side effect after ECT [17]. In our study, it was found that myalgia was statistically significantly more common in P group than in R group, considering the side effects. Myalgia was observed in 15 female patients and 3 male patients, wherein it was more common among females. Myalgia is often caused by motor activity during convulsions, muscle fasciculations due to the use of depolarizing muscle relaxants, or both. Therefore, administering low-dose depolarizing muscle relaxants or nondepolarizing muscle relaxants may reduce the risk of myalgia [18]. In our study, we can attribute the higher incidence of myalgia in P group to higher motor activity due to the fact that muscle relaxants were not used in the said group. We treated myalgia in a short period of time by administering 500 mg paracetamol oral tablets to the patients experiencing myalgia as a side effect after ECT. Werawatganon et al. compared the incidence of myalgia between patients who were given succinylcholine before ECT and patients who were planned to undergo surgery and received succinylcholine for induction. The study consisted of 50 patients, 25 of whom were treated with ECT and 25 of whom underwent surgery. Thiopental 4-5 mg/kg and succinylcholine 1 mg/kg were administered to all patients during induction of anesthesia. Although myoglobin levels were higher in patients who underwent surgery than in patients who underwent ECT, there was no statistically significant difference between the two groups in terms of myalgia [19]. In one study, the incidence of myalgia in the group that underwent surgery and used succinylcholine as a muscle relaxant was similar to that in patients who received succinylcholine for ECT, indicating that, apart from ECT, the main cause of myalgia was also succinylcholine that was used in both groups [20]. Similar to our study, there are studies involving the administration of ECT with sedation. However, in terms of side effects, we did not encounter any condition requiring treatment other than myalgia in any of the two groups. Tripathi et al. administered ECT with 0.5 mg/kg propofol to 49 patients, while 50 patients were administered ECT with the unmodified method. Anxiety was less common in the propofol group. In the mentioned study, patients in the unmodified group (patients that were not administered any medication) had anxiety, whereas patients in the propofol group had discomfort due to branula insertion and pain due to propofol injection [21]. Shah et al. administered ECT with 10 mg of diazepam I.V. to 46 patients and reported that this was an alternative to unmodified ECT. They reported that patients had intraoral bleeding (n=12), confusion (n=6) and myalgia (n=5) as side effects [22].

Nausea and vomiting after ECT were attributed to the anesthetics administered and headache occurring after ECT [23]. Propofol has an antiemetic effect at low doses (0.5-1 mg/kg) [24]. Hypertension was observed in 2 (5%) patients in R group and in 2 (5%) patients in P group. During ECT, systolic blood pressure can go up to 200 mm/hg. It usually returns to normal levels shortly after the convulsion ends without any treatment [23]. In our study, there was no statistically significant difference between the groups in terms of arrhythmia, hypertension, nausea-vomiting, and tachycardia. These side effects did not require medical treatment, since they were within the normal clinical range.

Studies have shown that preoxygenation and O2 insufflation during a convulsion reduces respiratory complications that can be encountered during and after induction [25]. In our study, there were no patients who had an oxygen saturation lower than 92% during convulsions and recovery, since we preoxygenated the patients with 80% O2 at 8 l/min for 5 minutes before the induction of anesthesia.

Conclusion

The administration of propofol as an anesthetic right before ECT without the addition of a muscle relaxant provided adequate and safe anesthesia with rapid recovery and minimum side effects except for transient postoperative myalgia in psychotic and bipolar patient groups that were planned to undergo ECT as part of their treatment.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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Conflict of interest

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